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Extensive C-terminal deletion in human immunodeficiency virus type 1 Env glycoprotein arising after long-term culture of chronically infected cells.

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Human immunodeficiency virus type 1 (HIV-1) chronically infected (CI) cell lines were established from HIV-1HIB/LAI-infected MT-4 cells that survived acute infection. The HIV env gene expressed in the two long-term cultured cell lines differed from that of the lines cultured for shorter periods, by coding for a glycoprotein gp 160 that had the C terminus deleted. One long-term cultured cell line, CI-17, was studied in detail. An insertion of a premature stop codon in the env gene caused about 90% of gp160 molecules to be truncated (gp160x), lacking both cytoplasmic and transmembrane domains; these species were secreted into the cell medium, and could form oligomers with other truncated gp160 molecules as well as with their normal counterparts. CI-17 cells constantly yielded high levels of viral protein and relatively low quantities of infectious virus, without cytopathicity. However, acute infection of fresh MT-4 cells with CI-17-derived virus led to cytopathicity, the rate of which as well as the Env glycoprotein pattern depended on multiplicity: (i) using an infection dose of 10(-4) ID50/cell, cells died 7 to 8 days post-infection with normal gp160 synthesis predominating; (ii) with 10(-2) ID50, gp160x was produced as early as 48 h post-infection and cell death was delayed. Predominant gp160x formation occurred again when new CI cell lines were obtained with CI-17-derived virus. Thus, two human immunodeficiency virus variants, a normal and a defective one, are persistently expressed in CI-17 cells. The other long-term cultured CI cell line also expressed gp160 with a similar (albeit slightly longer) deletion of a C-terminal region in most molecules, but the cell lines that were cultured for shorter periods did not. These results suggest that the emergence of HIV variants with a C-terminal deletion in the Env glycoprotein, which coexist with normal virus, may play a role in maintaining the long-term growth capacity and viability of CI cells.

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